

C-reactive Protein–Albumin Ratio: New Marker in Mortality Risk and Disease Severity in Pulmonary Thromboembolism

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Received: 24 April 2026; Accepted: 31 July 2026



Visual Abstract

ABSTRACT

Background: Pulmonary thromboembolism (PTE), with increasing incidence and decreasing mortality rate, is still a common cause of cardiovascular mortality today. The aim of this study was to evaluate the clinical relationship between disease severity and mortality risk and C-reactive protein (CRP)–albumin ratio (CAR) in patients admitted to the hospital with acute PTE.

Materials and methods: In this single-center, retrospective study, 215 patients diagnosed with PTE between 2012 and 2022 were included. All patients were divided into four groups according to mortality risk.

Results: A total of 47 patients were in the low-risk group, 91 patients were in the intermediate-low-risk group, 59 patients were in the intermediate-high-risk group, and 18 patients were in the high-risk group. There was a statistically significant difference between mortality risk groups in terms of CAR and other biochemical parameters ($p < 0.001$). A positive correlation between CAR and mortality risk, troponin, D-dimer, pro-brain natriuretic peptide (pro-BNP), simplified Pulmonary Embolism Severity Index (sPESI), and systolic pulmonary artery blood pressure (sPAB) parameters ($r_s = 0.440, p < 0.001$; $r_s = 0.277, p < 0.001$; $r_s = 0.215, p = 0.001$; $r_s = 0.236, p = 0.001$; $r_s = 0.318, p < 0.001$; $r_s = 0.201, p = 0.005$, respectively), and a negative correlation with tricuspid annular plane systolic excursion (TAPSE) ($r_s = -0.167, p < 0.001$) were found. In the receiver operating characteristic (ROC) analysis, CAR demonstrated a high discriminative ability in predicting high mortality risk, with an area under the curve (AUC) of 0.928.

Conclusion: This study shows that CAR may have clinical importance in PTE patients, helping to predict prognosis and guide treatment strategies. Its simplicity and cost-effectiveness may offer advantages in practice. However, further studies are needed to validate CAR in larger populations.

Keywords: C-reactive protein–albumin ratio, Inflammatory markers, Mortality risk, Prognostic biomarker, Pulmonary thromboembolism.

Journal of The Association of Physicians of India (2026); 10.59556/japi.74.1707

INTRODUCTION

Pulmonary thromboembolism (PTE) is still a common cause of cardiovascular mortality today, with increasing incidence and decreasing mortality.¹ It usually occurs as a complication of deep vein thrombosis (DVT) and is one of the most common acute cardiovascular diseases, accounting for approximately 5–10% of mortality in hospitalized patients.² The diagnosis of PTE begins with clinical suspicion; its symptoms are nonspecific and can be detected in a wide range of clinical presentations. Diagnosis can be made with a detailed evaluation that includes a combination of clinical symptoms, plasma D-dimer measurements, and imaging tests. Determining the mortality risk is very important for predicting the course of the disease and making treatment decisions. It is evaluated by combining the data on the hemodynamic status of the patient, the Pulmonary Embolism Severity Index (PESI) or Simplified PESI (sPESI) score, the involvement of the right heart, and the cardiac biomarker troponin value. Mortality risk is classified as high (massive), moderate (submassive), and

low (nonmassive). Intermediate-high-risk patients have right ventricular dysfunction and elevated troponin levels but intact hemodynamics and require close monitoring as their clinical condition may deteriorate at any time. In low-mortality-risk PTE, systemic blood pressure and right ventricular function are normal.¹

Serum biomarkers may also help determine the prognosis of the disease because inflammation accompanies thrombi. For this reason, as well as the algorithms mentioned above, simple and easily accessible laboratory methods are also practical. It is considered that acute-phase reactants may provide information about the severity of the disease in the early stages of embolism.

As a new parameter, C-reactive protein (CRP)–albumin ratio (CAR) provides better results compared with the use of these markers alone and is considered a marker associated with poor prognosis and mortality in various diseases.³ A retrospective study showed that CAR is an independent prognostic factor of PTE ($p = 0.03$).⁴ In the present study, our purpose was to

evaluate the clinical relationship between CAR and disease severity and mortality risk in patients with acute PTE. Also, we aimed to investigate whether CAR has prognostic value in determining the indication for escalation therapy (thrombolytic therapy) when hemodynamic deterioration occurs in some intermediate-high-risk patients. Furthermore, we investigated the relationship between CAR and troponin, D-dimer, pro-BNP, systolic pulmonary artery pressure (sPAP), and tricuspid annular plane systolic excursion (TAPSE).

PARTICIPANTS AND METHODS

This single-center, retrospective study was conducted in line with the principles of the Declaration of Helsinki after approval by the Gazi University Ethics Committee, numbered 2022-1465, and received on 30th December 2022. The study included 215 PTE patients who were followed up in our clinic between 1st December 2012, and 30th December 2022. The flowchart of the study is summarized in Figure 1. Patients who had inaccessible or missing data, concurrent acute coronary syndrome, hematological diseases that may lead to deterioration in laboratory parameters, terminal cancer, symptoms of acute infection, and pregnant women were excluded from the study. Demographic data of the patients (age, sex), smoking history (never smoked/quit/active smoker), concomitant diseases [diabetes, hypertension, heart diseases, chronic obstructive pulmonary disease (COPD), cancer, chronic renal failure, etc.], and PTE risk factors (immobilization, cancer, surgical history, etc.) were recorded. The

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How to cite this article: Orujlu A, Mammadova A, Yalçinkaya Z, et al. C-reactive Protein–Albumin Ratio: New Marker in Mortality Risk and Disease Severity in Pulmonary Thromboembolism. *J Assoc Physicians India* 2026;74(10):36–40.

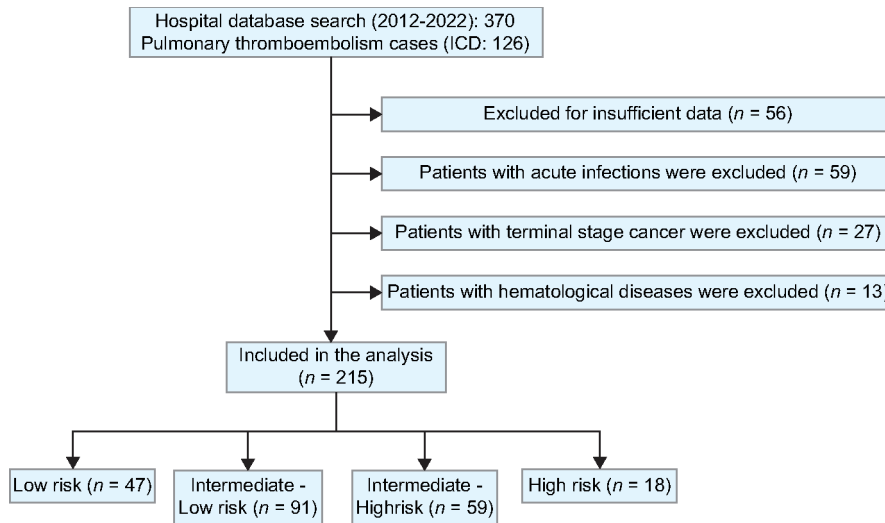


Fig. 1: Flowchart

sPESI score for PTE, hemogram, CRP, albumin, D-dimer, troponin, pro-BNP, CAR, pulmonary computed tomography angiography results, treatment status [thrombolytic therapy, heparin, direct oral anticoagulant (DOAC), warfarin], and echocardiography results (pulmonary artery pressure, right heart enlargement, TAPSE) were also recorded in the forms. All patients were divided into four groups based on mortality risk as low risk, intermediate-low risk, intermediate-high risk, and high risk. Mortality risk was calculated based on the hemodynamic status of the patients, sPESI score, cardiac troponin values, and echocardiography results, as recommended by current guidelines.¹ The sPESI was calculated based on the clinical parameters of the patients at the time of admission. If the patient had multiple records, the first laboratory parameter at the time of admission or suspicion of PTE was recorded. The CAR was obtained by calculating the ratio between these two biomarkers.

Statistical Analysis

Statistical analyses were performed using IBM SPSS version 23. Descriptive statistics were given as median (minimum and maximum values) and mean $\pm$ standard deviation for quantitative variables. Qualitative variables were expressed as frequency and percentage. To determine the normality of continuous variables, the Shapiro-Wilk test, histogram, and box plot results were evaluated. Since the normal distribution assumption was not met, the difference between the distributions of the two independent groups was compared with the Mann-Whitney *U* test. The Kruskal-Wallis analysis of variance was used because the normal distribution and variance homogeneity assumptions were not met for more than two independent groups.

In case of differences between the groups, pairwise comparisons were examined with the Dunn-Bonferroni test. For categorical variables, the difference between the groups was examined with the Chi-squared test or Fisher's exact test (or the extended version of this test, the Fisher-Freeman-Halton test). The relationship between two quantitative variables was examined with the Spearman correlation coefficient because the normal distribution assumption was not met. Receiver operating characteristic (ROC) analysis was used to examine the discrimination of the CAR between patients with acute PTE diagnosed with intermediate-high and high mortality risk, and the area under the curve (AUC) was evaluated. Also, the cutoff point to distinguish these two groups was determined by the Youden index. The significance level was set at 0.05 in all tests.

RESULTS

A total of 215 patients [124 (57.7%) female] were included in the study. The mean age of the patients was 62.9 ± 15.5 years. Demographic data and laboratory parameters of the patients are given in Table 1. A total of 47 patients (21.8%) had an sPESI score of 0. When the mortality risk group was examined, 18 (8.4%) patients were in the high-risk group, 59 (27.4%) were in the intermediate-high-risk group, 91 (42.3%) were in the intermediate-low-risk group, and 47 (21.9%) were in the low-risk group. No significant difference was detected between the mortality risk groups in terms of sex and age distribution ($p > 0.05$) (Table 2). The median CRP and CAR values of the whole group were 36 (2.68–198) and 11.0 (0.57–83.7), respectively. For mortality risk, statistically significant differences were found in CRP values between the low- and

Table 1: Demographic and laboratory characteristics of acute PTE patients

	Total number of patients (n = 215) n (%), med (min–max)
Comorbidities	
Hypertension	81 (41.1%)
Diabetes mellitus	43 (20.0%)
Coronary artery disease	19 (8.8%)
Congestive heart failure	27 (12.6%)
Chronic obstructive pulmonary disease	26 (12.1%)
Cerebrovascular accident	11 (5.1%)
Chronic renal failure	12 (5.6%)
Risk factors for PTE	
Deep vein thrombosis	44 (20.5%)
Malignancy	65 (30.2%)
Long term travel (>6 hours)	22 (10.2%)
Immobilization	58 (27.0%)
Presence of thrombophilia	10 (4.7%)
Bed rest (>3 days in the last 4 weeks)	51 (23.7%)
Estrogen use	11 (5.1%)
Operation (last 12 weeks)	45 (20.9%)
Trauma (last 12 weeks)	11 (5.1%)
Biochemical parameters of PTE patients	
CRP (mg/L)	36 (2.68–198)
Albumin (gm/dL)	3.67 (1.38–4.85)
Troponin T (ng/L)	16 (1–428)
D-dimer (ng/mL)	2.43 (0.47–35.2)
Pro-BNP (pg/mL)	385 (4.52–12500)
CAR	11.0(0.57–83.7)

CAR, CRP-albumin ratio; PTE, pulmonary thromboembolism

intermediate-low-risk ($p < 0.001$), low- and intermediate-high-risk ($p < 0.001$), low- and high-risk ($p < 0.001$), and intermediate-low- and high-risk ($p = 0.018$) groups, but no significant difference was observed between the intermediate-high- and high-risk groups ($p > 0.05$). Similarly, for CAR, significant differences were found between the low-risk and intermediate-low-risk ($p < 0.001$), low-risk and intermediate-high-risk ($p < 0.001$), low-risk and high-risk ($p = 0.001$), intermediate-low-risk and intermediate-high-risk ($p < 0.001$), and intermediate-low-risk and high-risk ($p = 0.008$) groups, but no significant difference was detected between the intermediate-high- and high-risk groups ($p > 0.05$) (Table 3 and Fig. 2).

The Spearman correlation analysis revealed a moderate, positive, and significant association between CAR and mortality risk ($r_s = 0.440$, $p < 0.001$). A low-level, positive,

significant association was detected between CAR and troponin ($r_s = 0.277$, $p < 0.001$), pro-BNP ($r_s = 0.236$, $p = 0.001$), D-dimer ($r_s = 0.215$, $p = 0.001$), sPESI ($r_s = 0.318$, $p < 0.001$), and sPAB ($r_s = 0.201$, $p = 0.005$). A negative, significant correlation was detected between CAR and TAPSE ($r_s = -0.167$, $p < 0.001$).

When the patients' CTPAs were reviewed, 85 (39.5%) patients were found to have a pulmonary infarction area. The median CRP and CAR values of patients who had infarction were found to be significantly higher than

those of patients who did not have infarction (CRP: 57.14 vs 28.1, $p = 0.004$; CAR: 16.8 vs 7.21, $p = 0.003$).

ROC analysis was performed to examine the role of CAR in distinguishing low- and high-mortality-risk patients. The threshold value was found to be 8.7, and according to this value, sensitivity was 89.2% and specificity was 83.6%. The area under the ROC curve was calculated as 0.928 (0.880–0.977), and this value was statistically significant ($p = 0.001$) (Fig. 3).

Among the 59 patients who had intermediate-high mortality risk, the mean CAR value in patients requiring escalation therapy (thrombolytic therapy) ($n = 6$) was 12.1 ± 9.01 . The rest of the intermediate-high-risk mortality patients (who did not require escalation therapy) had a mean CAR value of 20.4 ± 13.6 . It was detected that CAR did not have a distinctive role in predicting the need for thrombolytic therapy in intermediate-high-risk mortality patients ($p > 0.05$).

DISCUSSION

Acute PTE is the third most common cause of cardiovascular mortality due to its increasing incidence.⁵ In cases with high mortality and morbidity, it is important to make a rapid diagnosis and start treatment at an early stage due to the possibility of early reembolism. For this reason, risk stratification in patients diagnosed with PTE is essential both to predict the prognosis of the disease and to guide treatment decisions. Despite current mortality risk assessments, simple and easily accessible methods are still needed to predict the prognosis of the disease.⁶ In the present study, the role of CAR was investigated in

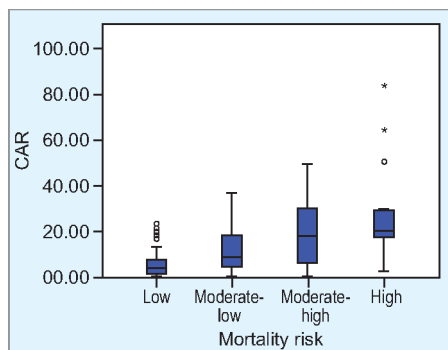


Fig. 2: Distribution of CAR values in different mortality risk groups

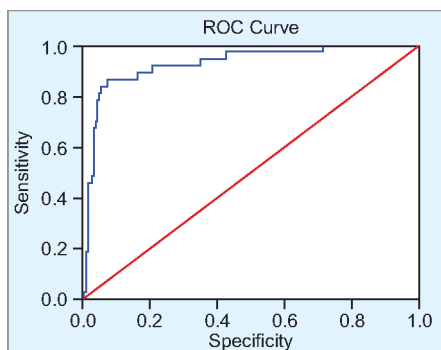


Fig. 3: ROC curve of CAR to distinguish low- and high-risk patients

Table 2: Demographic and disease-related characteristics of patients who had acute PTE according to mortality groups

	Low risk $n = 47$	Moderate-low risk $n = 91$	Moderate-high risk $n = 59$	High risk $n = 18$
Age (mean $\pm$ SD)	54.8 $\pm$ 19.1	63.6 $\pm$ 13.7	69.4 $\pm$ 12.3	59.3 $\pm$ 12.9
Sex (n) (%)				
Male	24 (51.1%)	37 (40.7%)	23 (39%)	7 (38.9%)
Female	23 (48.9%)	54 (59.3%)	36 (61%)	11 (61.1%)
Smoking status				
Active smoker	25 (53.2%)	37 (40.7%)	15 (25.4%)	4 (22.2%)
Never smoked	22 (46.8%)	54 (59.3%)	44 (74.6%)	14 (77.8%)
Hospitalization status				
Inpatient treatment	40 (85.1%)	86 (94.5%)	59 (100%)	18 (100%)
Outpatient treatment	7 (14.9%)	5 (5.5%)	0	0
History of PTE	4 (8.5%)	9 (9.9%)	10 (16.9%)	0

PTE, pulmonary thromboembolism

Table 3: Laboratory and echocardiographic values among mortality risk groups

	Low risk Med (min–max)	Moderate risk Med (min–max)		High risk Med (min–max)	p -value
		Low risk	High risk		
CRP	18 (3–106)	34 (2.68–114)	60 (3–134)	68.9 (22–198)	$p < 0.001$
Albumin	3.9 (1.38–4.85)	3.7 (2.4–4.7)	3.5 (2.8–4.5)	3.08 (1.85–4)	$p < 0.001$
CAR	4.44 (0.57–23.8)	11.5 (0.6–36.8)	16.47 (0.91–49.44)	21.6 (10.33–83.78)	$p < 0.001$
D-dimer	1.26 (0.6–9.12)	2.43 (0.47–13.9)	4.41 (0.55–20)	3.51 (1.2–35.2)	$p < 0.001$
Pro-BNP	142 (9–2481)	242 (4.52–7877)	1752 (10–12500)	2490 (66–10000)	$p < 0.001$
Troponin	8.0 (1.0–54)	11 (1.0–262)	45 (1.0–428)	147 (34–288)	$p < 0.001$
PAP (mm Hg)	25 (20–29)	28 (20–58)	54 (30–79)	60 (45–91)	$p < 0.001$
TAPSE (mm)	22 (20–34)	22 (14–27)	17 (10–23)	17 (9–24)	$p < 0.001$

CAR, CRP–albumin ratio; CRP, C-reactive protein; ECHO, echocardiography; PAP, pulmonary artery pressure; TAPSE, tricuspid annular plane systolic excursion

predicting mortality risk and determining disease severity in PTE patients.

We found the infarction frequency to be 39.5%, and this result is parallel to the results in the literature. In another study in the literature, the pulmonary infarction rate was reported as 29.2% in 154 patients diagnosed with PTE.⁷ In a previous review, it was reported that pulmonary infarction was seen in 9–36% of patients in various studies performed with the CTPA method.⁸ In a single-center, retrospective study conducted by Yuan et al. in China, it was found that 16.58% of 386 PTE patients had infarction, and CRP >10 mg/L was detected in 65% of patients who had infarction. This result was significantly higher than that in patients who did not have infarction ($p < 0.001$).⁹ Also, in a different study, CRP levels of PTE patients who had infarction were found to be significantly higher compared with those of patients who did not have infarction ($p < 0.0003$).¹⁰ Although the relationship between CRP and pulmonary infarction was evaluated in detail in the studies mentioned above, to the best of our knowledge, the relationship between CAR and infarction has not yet been examined in the literature. In our study, we observed that CRP and CAR values of patients who had infarction were significantly higher compared with those of patients who did not have infarction ($p = 0.004$, $p = 0.003$).

The acute-phase response includes a series of inflammatory processes that occur in the body as a response to infection, tissue damage, and immunological disorders. CRP is a positive acute-phase reactant synthesized by the liver, and its blood level is elevated within hours as a response to inflammation and infection. In particular, elevated levels of CRP were associated with a series of diseases such as infections, tissue damage, inflammatory conditions such as psoriatic arthritis, VTE, and cardiovascular events.^{11,12} CRP increases tissue factor synthesis, activates homeostasis, and can affect fibrinolysis, which is considered to be one of the mechanisms in the development of VTE.¹³ Studies were conducted for many years showing the relationships between CRP and VTE, and the Reasons for Geographic and Racial Differences in Stroke (REGARDS) cohort showed that there was a positive relationship between CRP and VTE and that the risk of VTE increased with high CRP.¹⁴ In the study conducted by Elshahaat et al., patients were divided into low-, moderate-, and high-mortality-risk groups, and a statistically significant increase was observed among all groups in terms of CRP.⁶ Similarly, another study found significant differences in CRP levels in the same risk groups.¹⁵ We found that there was

a statistically significant difference between mortality risk groups in terms of CRP, similar to the literature. When mortality subgroups were evaluated, only the CRP values of the intermediate-high- and high-risk groups were similar. This can be interpreted as indicating that patients in the intermediate-high-risk group must be closely monitored and that these patients may develop a high mortality risk at any time.

As a negative acute-phase reactant, serum albumin decreases as a response to inflammation.¹⁶ The REGARDS cohort also showed that low albumin levels increase the risk of VTE.¹⁴ In the study conducted by Sağcan et al., serum albumin levels in patients who had high-mortality risk were found to be significantly lower than those in patients who had low-mortality risk ($p = 0.031$).¹⁵ Here, we showed that there was a significant difference between the high- and intermediate-high-risk groups and the low-risk group in terms of albumin, similar to the literature.

In a retrospective study that examined the prognostic value of CAR in acute PTE, 186 patients were examined, and it was reported that although it had lower sensitivity than the PESI score in predicting mortality risk, it had similar specificity.¹⁷ In a retrospective study by Hocalı and Tanrıverdi, CAR was an independent predictor in PTE patients with high-mortality risk,¹⁸ and in another study, CAR was shown to serve as a prognostic marker in predicting all-cause mortality in PTE patients both at 30 days and in the late period.¹⁹ It is a strong biomarker in determining prognosis in different types of cancer, such as esophageal cancer and upper gastrointestinal (GI) cancers, and a high CAR value is associated with poor prognosis in gastric cancer patients and can predict complications after gastrectomy.^{20,21}

The results of the present study showed that CAR is significantly different among mortality risk groups and that there was a moderate positive relationship between CAR and mortality risk in the correlation analysis. In line with the literature, it was shown that as CAR increases, the mortality risk increases; for this reason, CAR is a prognostic marker that can be obtained easily and quickly in determining mortality risk and does not require any additional cost.

In the study conducted by Özcan et al., it was reported that CAR, troponin, and PESI scores showed a moderate positive correlation.¹⁷ They reported that a threshold value of 5.33 for CAR predicted 6-month mortality with 65.3% sensitivity and 65.6% specificity,¹⁷ while another study showed that CAR was an independent predictor of late-term mortality and was more effective than both albumin and CRP.²² Since our study

was a retrospective screening and mortality information of all patients could not be obtained, we could not examine the role of CAR in determining mortality. However, when a threshold value of 8.7 was used for CAR, sensitivity and specificity were found to be 89.2 and 83.6%, respectively, in predicting the high-risk group. In the correlation analysis of the present study, a positive correlation was detected between CAR and troponin, pro-BNP, D-dimer, sPESI, and sPAB.

CONCLUSION

The results of the present study show that patients who have high CAR have a significantly increased mortality risk and a more severe disease course. Also, the positive correlation of this ratio with biomarkers such as troponin, sPESI, and pro-BNP emphasizes the clinical importance of using it as a prognostic indicator in determining the course of the disease in PTE patients.

In particular, the CAR may be a valuable tool in emergency departments where access to advanced diagnostic modalities is limited. In centers where echocardiography and cardiac biomarkers such as troponin are not readily available, CAR, based on simple, inexpensive, and rapidly obtainable laboratory tests, could serve as a surrogate marker to assist in early risk stratification of patients with suspected pulmonary embolism. Integrating CAR into the initial clinical evaluation may aid clinicians in identifying patients at higher risk who require urgent referral or closer monitoring, especially in resource-constrained settings. While it is not a replacement for a comprehensive assessment, CAR may support decision-making when key parameters of established prognostic models are not accessible during presentation.

The fact that CAR is simple, economical, and widely available may increase the clinical applicability of this parameter. However, the results of the present study showed that it has no significant role in determining escalation therapy in patients who have intermediate-high mortality risk when clinical deterioration occurs.

In conclusion, the present study highlights the potential role of CAR in determining mortality risk and disease severity in patients who had PTE, indicating that it may be a valuable prognostic indicator that can be used in clinical practice.

Limitations

The present study had several limitations. First, it had a retrospective design and

relied on archival data. The lack of a prospective controlled study may prevent us from understanding the cause-and-effect relationships more clearly. Second, the small number of patients who had high-risk and intermediate-high-risk disease and who had worsening hemodynamics during follow-up may not have fully represented these groups. With these limitations in mind, the results must be carefully evaluated and interpreted, emphasizing the need for larger and more comprehensive studies to understand the prognostic value of CAR in PTE patients.

ETHICAL DECLARATIONS

The study was approved by the Gazi University Clinical Research Ethics Committee (Date: 30th December 2022; Decision No.: 1465).

DECLARATION OF AUTHORSHIP

All authors conceived and designed the study; AO and AM acquired the data; AO, AM, and ZY analyzed and interpreted the data; AO and İKO drafted the manuscript; AM and İKO critically reviewed the manuscript for important intellectual content; all authors gave approval of the version to be submitted; and all authors agreed to be accountable for all aspects of the work.

HIGHLIGHTS

The CAR is a significant prognostic marker for mortality risk and disease severity in patients with acute PTE.

Elevated CAR levels are positively correlated with increased mortality risk and worse clinical outcomes.

CAR shows significant positive correlations with established cardiac and clinical biomarkers such as troponin, pro-BNP, and the simplified Pulmonary Embolism Severity Index.

Both CRP and CAR levels are significantly higher in patients presenting with pulmonary infarction compared with those without infarction.

CAR, as a simple, cost-effective, and rapidly obtainable laboratory test, may serve as a valuable tool for early risk stratification in emergency settings with limited access to advanced diagnostic modalities.

AUTHOR CONTRIBUTIONS

AO, AM, and İKO designed and coordinated the study, participated in data acquisition and interpretation, and drafted the article. AO and AM participated in data collection. Statistical analyses were performed by ZY and reviewed in detail by İKO. All authors participated in the review and revision of the manuscript. All authors have approved and take responsibility for the final version of the manuscript.

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